

## Personalised diet

### PERSONALIZED ANTI-INFLAMMATORY NUTRITION FOR ATOPIC ECZEMA AND PSORIASIS PATIENTS

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Our experience in the treatment of over 20,000 atopic eczema and psoriasis patients shows that besides allergic reactions to foods an increasing number of pseudoallergic reactions caused by toxic-irritative pollutants (formaldehyde, exhaust particles, food additives, nicotine, wood preservatives, pesticides, heavy metals) are responsible for the inflammatory process behind the complex symptoms. Intrauterine and postnatal environmental influences were also reported.

The routine analysis of specific IgE- and IgG4-factors in atopic patients by means of ELISA-assays after challenge meals (CM) showed an increased frequency of specific IgE- and IgG4-antibodies after repeated CM. In 60% of the patients, we simultaneously found raised concentrations of circulating immune complexes with food specific IgE- and IgG ( $p<0.005$ ) responsible for the delayed (Type III) allergic reactions in the same group.

Further inflammation markers like acute phase proteins ( $\alpha$ 1-antitrypsin,  $\alpha$ 2-makroglobulin, haptoglobin and caeruloplasmin) showed a surprisingly rapid increase after CM ( $p<0.01$ ) in the atopic group, by contrast the control samples remained in the normal range.

Serum histamine levels (RIA-Test) also showed a significant increase 1/2 h after CM and after individual oral provocation with lactose, sucrose, tyramine, serotonin or phenylethylamine, respectively. The carbohydrate intolerance reactions (H2-test) were in good correlation to the significantly lowered disaccharidase activities (lactase, sucrase) in the gut of atopic eczema and psoriasis patients ( $p<0.001$ ). This was closely related to

chronic intestinal dysbiosis with toxic microbial breakdown products (alcohols, aldehydes, phenols, diamines) leading to an increased intestinal permeability, histamine release and impairment of liver detox function.

On the other side, we found both in atopic eczema and in psoriasis patients pseudoallergic reactions against biogenic amines and constantly raised serum histamine levels (also in fasted patients), suggesting an inhibition of catabolic enzymes (MAO, DAO, NMT). Previously published results from our laboratory showed significantly reduced DAO ( $p<0.001$ ) and Type B MAO- ( $p<0.05$ ) activities in thrombocyte rich plasma of atopic eczema and psoriasis patients explaining their intolerance reactions to histamine, tyramine and octopamine rich foods.

Last, but not least, the chronic increased levels of free radicals in whole blood and plasma of all patient groups showed significant changes after oral intake of different food homogenates/juices depending on their ROS-blocking or ROS-increasing effects (enhanced chemiluminescence test).

Our current research shows that appropriate combinations of hypoallergenic protein hydrolysates with selected sugar alcohols and omega-3 fatty acids are dramatically enhancing the anti-inflammatory and free radical quenching properties of the mixture, demonstrating an obvious therapy effect in atopic and psoriasis patients.

With the emergence of affordable microarray gene expression profiling methods, the opportunity arises to ex-vivo test certain nutraceutical combinations for their silencing ability on inflammatory genes before and after their administration to the patient (nutrigenomic evaluation).

The input of the above nutritional data in a computer programme (FOOD ALLERGY CONTROL®) help us generate personalized rotation diet plans that ensure a rapid symptom improvement and stabilization of the clinical course in both groups of patients.

## Lecture objectives

1. To recognize the crucial role of toxic-irritative pollutants (pesticides, wood preservatives, food additives, solvents, heavy metals, nicotine, exhaust particles, etc.) and chronic infections as triggers of pseudoallergic reactions and obligate cofactors of allergic answers to food and respiratory allergens, respectively.
2. To understand the role of the above mentioned agents and offending food in the free radical generation process with subsequent activation of transcription families and activation of pro-inflammatory genes by means of appropriate gene expression profiling methods.
3. To identify main players of the immune and inflammatory response to food and food additives like specific antibodies of IgE and IgG classes, acute phase proteins, interleukins and other cytokines, prostaglandins, leukotrienes, adhesion and coagulation factors.
4. To understand the specific food selection needed to counteract the free radical generation and the inflammation cascade in skin and atopic patients through individualized diets.

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## WOMEN'S HIGHER HEALTH RISK WITH INCREASED N-6 PUFA INTAKE COMPARED TO MEN: AN 'ISRAELI GENDER PARADOX' HYPOTHESIS

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Israeli health status was found to be lower than in most other Mediterranean countries, despite the 'good' Israeli diet—low in calories and fat, and high in ratio of polyunsaturated fatty acids (PUFA) to saturated fatty acids (SFA), fruits, and vegetables. This unexpected dissociation was defined as the 'Israeli paradox' and attributed to high consumption of n-6 PUFA. However, gender analysis showed that where women ranked unexpectedly low, i.e. 11th/15 European countries in life expectancy, whereas men ranked among highest. This could suggest a gender differential in health profiles with the same diet, which appears to be concentrated mostly in cancer risk.

Trends of gender-specific risk were also observed in comparisons of ethnic subgroups. Jewish women have had much higher cancer prevalence (1:3) than Israeli-Arab women (1:6) and much higher cancer mortality, with higher dietary n-6 PUFA (mostly linoleic acid from corn and soy oil), compared to n-9 monounsaturated fatty acids (MUFA, mostly oleic acid from olive oil). However, Israeli-Arab women recently have had a significantly faster-growing rate, where men have remained relatively stable. This is concurrent with dietary westernization, including exchanging traditional olive oil for high n-6 PUFA oils (mostly soy), resulting in increased n-6 PUFA consumption and high dietary n-6:n-9 and n-6:n-3 FA ratios.

Experimental and clinical research have suggested sexual dimorphism in lipid and PUFA metabolism and potential carcinogenicity that may support the assumption regarding women's higher risk with a high n-6 PUFA diet. This can be further adjunctive to genetic predisposition, as well as to loss of the protective effects of olive oil against breast cancer mutations.

Conclusions: This is the first time sexual dimorphism has been suggested as a modulating factor of dietary effects on public health and a national shift in disease epidemiology, linking dietary 'de-Mediterraneanization' and high n-6 PUFA consumption to western health risks. Supportive evidence suggests that gender and/or genetic predisposition, previously suggested to be the explanatory factor for high Israeli women's cancer risk, do not preclude a causal role of nutrition and its preventive potential, but rather emphasizes its importance.

## PREDICTIVE DIAGNOSTIC VALUE OF BIOMARKERS FOR RENAL FAILURE IN MEDICAL EMERGENCY RELATIONSHIP OF NGAL (NEUTROPHIL GELATINASE ASSOCIATED LIPOCALIN) WITH UNDERWEIGHTNESS AND MALNUTRITION

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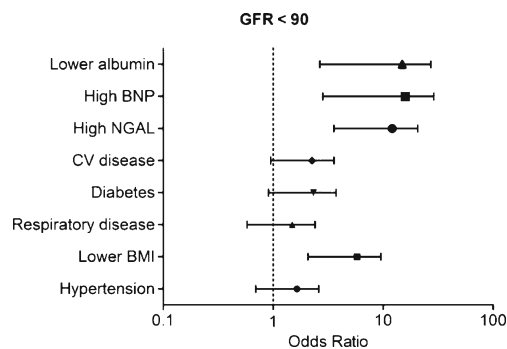
Among the new biomarkers of acute kidney injury, NGAL (neutrophil gelatinase-associated lipocalin) seems one of the most useful even in the emergency room. Nonetheless, its predictive value for severe acute kidney injury (AKI) is still uncertain. The higher frequency of acute kidney injury in patients arriving in the emergency department is one of the main factors that lead to an increase of length of hospitalization and mortality and hence to more prolonged and higher health care costs. Although there are enough validated criteria for the definition of the stages of renal damage (AKIN classification, stages 1–3) or with the RIFLE criteria (that define five classes), there are no reliable criteria of predictability and risk. Despite NGAL thresholds' predictor for subsequent acute kidney injury have been defined in various disease, unfortunately studies that define the actual risk in the subset of baseline normal renal function values are still few.

The aim of this study is the assessment, in subjects without a previous history of chronic renal failure, of the degree of risk of renal injury-failure associated with abnormal baseline NGAL.

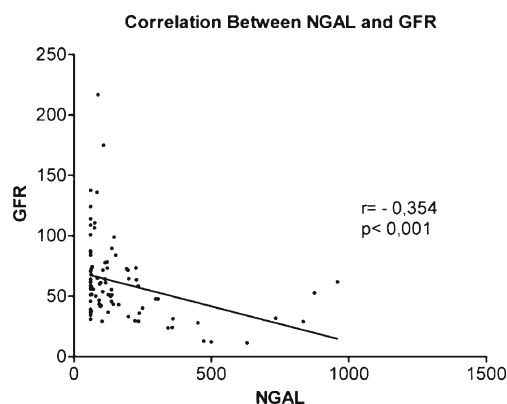
**Patients and methods:** We studied 115 patients (M 64, D 51), mean age  $74.71 \pm 11.07$  years that, at the time of presentation-triage in the emergency department, were classified as maximum or high priority for critical respiratory events and dyspnea. Patients with actual or previous chronic renal failure ( $GFR > 90$ ), i.e. before the current admission, patients with cancer, obesity ( $BMI > 30$ ), moderate to severe anemia ( $Hb < 11$  g/dl) and fever—body temperature  $> 38.5^\circ\text{C}$  were excluded. All patients were followed with clinical monitoring, routine emergency blood tests, continuous blood pressure and ECG monitoring. Chest x-ray and echocardiograms were performed at the admission, and subsequently, whenever indicated. Baseline serum NGAL was assayed, and was thereafter checked at 6, 12, 24, 48 and 72 h.

**Statistical analysis:** the degree of risk (Odds ratio) for acute kidney injury was defined as a GFR decrease below 60; correlation analysis vs. GFR changes of NGAL, BMI, BNP, age and serum albumin were performed individually. MLR models were used in order to assess predictability of GFR decrease and of renal failure.

**Results:** The Odds Ratio demonstrates an increased risk of kidney injury in patients with higher NGAL (Odds ratio 6.085, 95% CI 1.282 to 28.883), lower BMI (odds ratio, 3.401; 95% CI 0.883 to 13.108) and serum albumin below the normal values, and in patients with higher BNP.



A significant direct correlation between NGAL and GFR was observed as well.



By the MLR model, using the baseline measurements, GFR decrease is explained by NGAL, BMI, Albumin and BNP.

**Table Stepwise Linear Regressions to GFR**

	<i>GFR T6</i>					
	<i>R</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>sig</i>	$\beta$	<i>p</i>
	0,679	0,461	16,249	<b>&lt;0,0001</b>		
BMI					0,508	<b>&lt;0,0001</b>
NGAL Time 0					-0,289	<b>0,003</b>

BMI: Body Mass Index; The complete predictive model includes BMI, NGAL, Diabetes, Systolic and Diastolic Blood Pressure Weighted Least Squares Regression—Weighted by Age

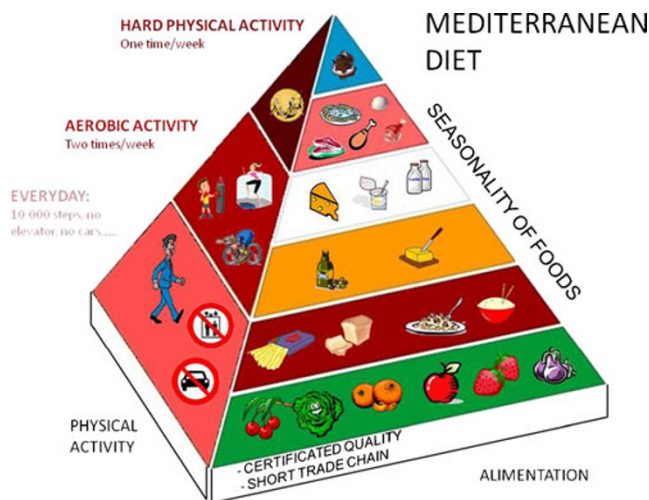
**Conclusions.** Considering our results NGAL is confirmed to be a valuable biomarker for acute kidney injury in patients admitted to the emergency department. The additional information provided by our study implies also a significant prognostic power of concurrent measurements related to malnutrition, and notably low albumin and underweightness.

## TAILORED MEDICAL TREATMENTS IN OBESITY. ADHERENCE TO THE MEDITERRANEAN DIET PROMOTE A LOWER INFLAMMATORY PROFILE AND MINOR INSULIN RESISTANCE AND LIPID METABOLIC ALTERATIONS

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The Mediterranean diet, due to its association with low morbidity and mortality for several chronic diseases, has been recognized as a model of healthy eating. Epidemiological studies and clinical interventions have shown an effect of the Mediterranean Diet on metabolic and inflammatory biomarkers. Therefore, it is conceivable that the protective role of diet on these might be mediated by two pathways involving concurrently inflammation and metabolic abnormalities. Purpose of the study is to assess whether increased adherence to Mediterranean Diet in obesity is associated with different clinical profiles in relation to inflammation and metabolic aspects.



**Patients and methods:** We studied 208 patients (139 F and 69 M), overweight or obese, age  $46.50 \pm 12.70$ , BMI  $30.27 \pm 4.46$ , referred for nutritional counseling. Exclusion criteria: therapy-dependent diabetes, infectious diseases or cancer, rheumatic diseases, inflammatory-granulomatous and autoimmune diseases, renal insufficiency (GFR  $<90$  ml/min), thyroid diseases. Mediterranean diet adherence was assessed through one-week/one year Dietary Recall Interview carried out by dietitians and taking into account the criteria of the Mediterranean Diet Score (AMDS: range 0–55). Subsequent dietary treatment was

accomplished by dietary prescriptions developed using a specific software (Dietosystem®).

**Results:** A negative correlation between AMDS and CRP ( $r = -0.312$ ,  $P < 0.0001$ ) was observed. Moreover, an inverse correlation with serum lipids was also found: Patients who have a greater adherence to a Mediterranean diet have lower levels total cholesterol ( $r = -0.261$ ,  $P < 0.0001$ ), triglycerides ( $r = -0.158$ ,  $P = 0.023$ ) and LDL cholesterol ( $r = 0.271$ ,  $P < 0.0001$ ).

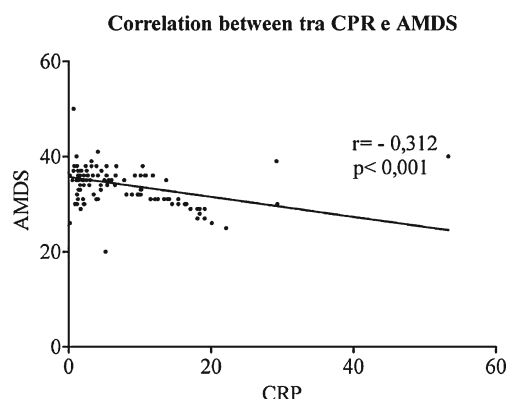


Table Multiple Linear Regressions to CRP and HOMA

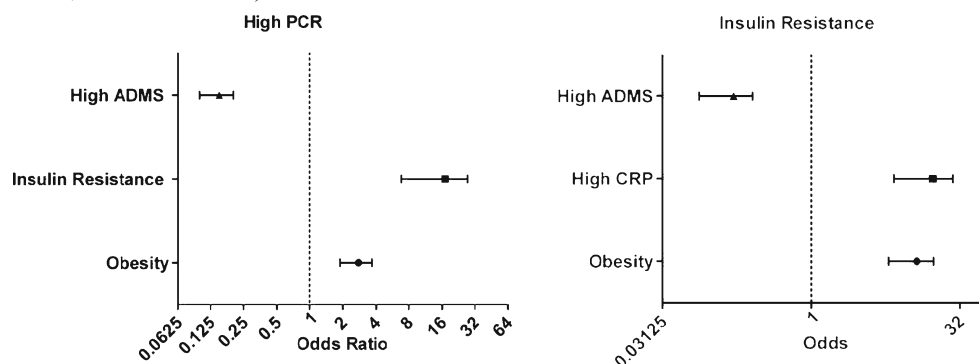
	CRP					
	R	R <sup>2</sup>	F	sig	$\beta$	p
BMI, Kg/m <sup>2</sup>	0,465	0,216	10,996	<0,0001	0,229	<0,001
HOMA					0,164	0,013
Total Cholesterol, mg/dl					0,000	0,994
Triglycerides, mg/dl					0,076	0,276
AMDS					-0,289	<0,0001
	HOMA					
	R	R <sup>2</sup>	F	sig	$\beta$	p
BMI, Kg/m <sup>2</sup>	0,340	0,116	5,216	<0,0001	0,127	0,084
Total Cholesterol, mg/dl					-0,069	0,331
Triglycerides, mg/dl					0,175	0,017
AMDS					0,029	0,689
CRP, mg/L					0,185	0,013

BMI: Body Mass Index; CRP: C Reactive Protein, AMDS: Adherence Mediterranean Diet Score.

The Odds ratio also shows that high levels of adherence to Mediterranean diet is associated with lower values of CRP



(OR: 0.132, CI 0.071–0.248), and insulin resistance (OR: 0.115, CI 0.039–0.338).



**Conclusions:** Our study suggests that adherence to traditional Mediterranean diet is associated with lower concentrations of at least one marker of inflammation and lipid risk factors in subjects without significant cardiovascular disease and without diabetes. This may partly explain the beneficial effects of this dietary profile on various chronic diseases, and confirms its probable role in secondary prevention. The use of Mediterranean Diet is reasonably to be advised and warranted also in obese subjects, due to its association with a favorable inflammatory and lipid metabolic profile, and lower insulin resistance.

#### PERSONALIZED NUTRITIONAL APPROACH AS THE CORE TREATMENT FOR NAFLD. GREATER ADHERENCE TO MEDITERRANEAN DIET IS A TARGETED PREVENTION

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NAFLD (Non-Alcoholic-Fatty Liver—Disease), because of its association with obesity, diabetes and insulin resistance (IR), can be considered the hepatic expression of metabolic syndrome. Mediterranean Diet is commonly recognized as a behavioral, clinical and epidemiological paradigm of healthy lifestyle.

The aim of this study is to assess relationship between adherence to Mediterranean diet score (AMDS) and NAFLD, a condition secondary to unhealthy eating habits, reduced physical activity and genetic factors.

**Methods:** We studied 274 consecutive subjects (160 F, 114 M) with a mean age of  $46.21 \pm 12.88$  years and BMI  $30.33 \pm 5.98$ , referred to our DH for nutritional counseling. Exclusion criteria were the presence of viral hepatitis, toxic or autoimmune disorders, alcohol abuse (less than 20 ml/day), diabetes, use of

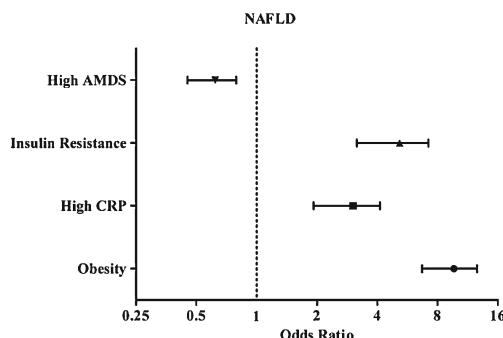
hepatotoxic drugs, insulin resistance. This last was evaluated by HOMA (Homeostasis Model—Assessment) and adherence to Mediterranean Diet was assessed by a validated score (AMDS: range: 0–55). Patients underwent ultrasonography of the upper abdomen and severity of steatosis was assessed by the Bright Liver Score (BLS). Dietary prescriptions were given using a proprietary software—Dietosystem®—that allows personalized recipes and lifestyle-physical activity counseling. **Results:** Patients were 133 NAFLD and 141 non-NAFLD; these were the control group, matched for age, sex and physical activity (Baecke Questionnaire). A lower adherence to the Mediterranean diet has been observed among patients with NAFLD compared with controls ( $33, 51 \pm 3.82$  vs.  $35.62 \pm 4.42$ ;  $p < 0.0001$ ). The two groups also differ for BMI ( $33.31 \pm 5.32$  vs.  $27.55 \pm 5.21$ ,  $P < 0.0001$ ) and HOMA ( $4.33 \pm 3.85$  vs.  $2.03 \pm 0.90$ ,  $p < 0.0001$ ) that are higher in NAFLD subjects. There is an inverse correlation between BLS and AMDS ( $r = -0.175$ ,  $p: 0.004$ ). In the linear regression model, weighted by gender (AMDS lower in women), BMI ( $p < 0.0001$ ), HOMA ( $p: 0.0004$ ) and AMDS ( $p < 0.0001$ ) explained together 55.1% of variance to BLS.

#### Multiple Linear Regressions to BLS

Predictors	R	R <sup>2</sup>	F	Sig	β	p
	0,742	0,551	17,677	<0,0001		
<b>BMI, Kg/m<sup>2</sup></b>					0,463	<0,0001
<b>HOMA</b>					0,291	0,004
<b>AMDS</b>					−0,354	<0,0001
<b>PCR, mg/L</b>					0,067	0,427
<b>AGE, y</b>					0,082	0,312

The Odds ratio shows that the risk of more severe liver steatosis is increased by the greater degree of obesity (OR: 8.747, CI: 5.051 to 15.147), by higher values of CRP (OR 2.624, CI:

1.350 to 5.101) and HOMA (OR: 4.392, CI: 2.143 to 9.001), and decreased by 50% from a greater degree of adherence to a Mediterranean diet (OR: 0.575, CI: 0.354 to 0.835).



**Conclusion:** Greater adherence to Mediterranean diet is associated with lower prevalence and severity of fatty liver. Exercise and nutritional profiles consistent with the Mediterranean diet, as in the case of the current guidelines, may prevent this and other related conditions. A reasonable therapeutic approach must include advices addressed at qualitative dietary changes and focused at strategies targeted for appropriate behavioral changes.

#### AD36 HUMAN ADENOVIRUS SEROPOSITIVITY. A NOVEL OBESITY BIOMARKER ASSOCIATED WITH ENHANCED RESPONSE TO DIETARY INTERVENTIONS

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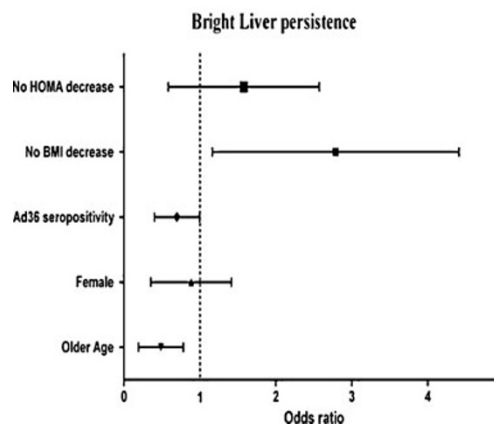
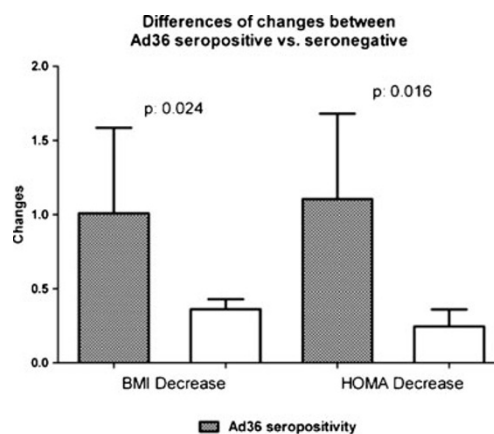
Obesity and related or consequent disease are both currently attributed to inappropriate lifestyle and nutrition. Higher prevalence of human adenovirus Ad36 seropositivity (Ad36+) is reported in obesity. This observation is mainly epidemiological and the predictability of obesity on the basis of Ad36 seropositivity is only conjectural. The aim of our study is to investigate whether lifestyle-nutritional intervention achieve different outcomes in obese patients, i.e. if they are blunted or enhanced according to Ad36 seropositivity status. Patients and methods: One-year nutritional intervention was planned and accomplished for 62 overweight-obese patients, studied by BIA and anthropometric nutritional assessment, homeostatic model assessment of Insulin Resistance (HOMA), and Ad36+ assay. Lower salt/lower calories Mediterranean diet, physical activity increase, smoking withdrawal and lifestyle counselling, contributed by the support of an health psychologist, were provided. Subjects attended a total of twenty scheduled individual encounters and visits with one of the physicians, the dietician and the psychologist for clinical evaluation during the first

6 months (1st–6th month) of follow-up. Suggestions and advice on individual “healthy” food purchase, storage and cooking are provided. Physical activity is encouraged mainly in the form of walking using the ‘10,000 steps a day’ suggestion, and adherence is also empowered by providing a portable electronic pedometer as a motivational and control tool.

**Results:** Ad36 seropositive patients have baseline greater BMI in comparison with Ad36 seronegative patients. Different prevalence of post-interventional response, that is significantly greater among Ad36+ patients, is observed: there was a greater decrease of obesity, assessed by BMI, and a greater reduction of insulin resistance, assessed by HOMA. By Odds ratio, Ad36 seropositivity is associated with a positive response to the nutritional intervention, i.e. higher prevalence of favorable response to diet.

In Ad36 seronegative NAFLD patients, whose metabolic profile, and notably, HOMA, improves without significant weight loss, an effect of dietary changes profile and not only of lowered food caloric intake is conceivably operating.

A BMI adjusted Multiple-Linear-Regression model explains significantly 23.8% ( $p < 0.04$ ) of the variance to significant



weight loss and to lowering of HOMA, with the significant contribution of Ad36 seropositivity.

Conclusion: Ad36+ obese patients, under dietary treatment, have a more consistent decrease of Insulin resistance and body weight, indicating a greater responsiveness to the nutritional intervention. Even if this can be due to the initial greater obesity of Ad36 seropositive patients, the subsequent Multiple Linear Regression model, BMI adjusted for eliminating the difference of BMI as a potential confounding factor, confirms the seemingly beneficial relevance of Ad36 seropositivity. This is not dependent on a greater pre-interventional body weight. Ad36 previous infection seems to enhance weight loss, and recovery of insulin sensitivity under nutritional interventional treatment.

### A PREDICTIVE DIAGNOSTIC SEROLOGICAL BIOMARKER OF NAFLD AD37 HUMAN ADENOVIRUS AS A PUTATIVE CAUSATIVE FACTOR

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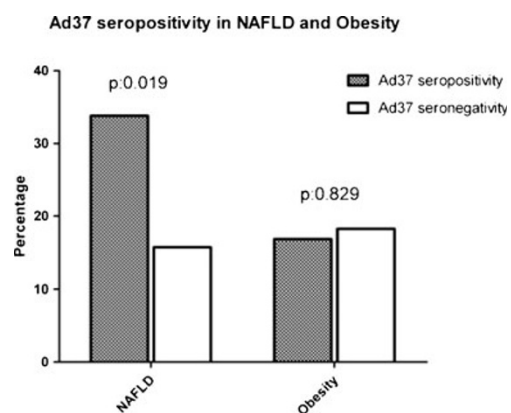
A significant association of Ad-36 seropositivity with obesity, with Non-Alcoholic-Fatty-Liver Disease (NAFLD) and with essential Hypertension in human beings is reported, but pathophysiology is still elusive. Ad-36 adipogenic adenovirus effect cannot be directly explained by an insulin-resistance-related mechanism and in adolescents Ad-36 seropositivity is strongly associated with lipid disorders regardless of obesity. A different Adenovirus, Ad-37, increases adiposity in animals.

NAFLD is a condition whose natural history is related to, but not completely explained by over-nutrition, obesity and insulin resistance. Its main feature is bright liver, an Ultrasound pattern that encompasses mostly fatty liver disease, irrespective of the actual causes. This condition is currently assessed non-invasively and ultrasound bright liver pattern and the derived score have a satisfactory clinical consistency. Insulin Resistance, commonly evaluated as HOMA, according to the Homoeostasis Model Assessment of Insulin Resistance index (HOMA or HOMA-IR), is a pathophysiological clue to several metabolic-dependent conditions, including obesity, hypertension, hyperlipidemia and type 2

diabetes. Despite increased highly sensitive C-reactive protein (CRP) assay was found to be associated with NAFLD and that it was suggested the use of CRP as a sensitive independent marker to detect fatty liver in the general population, a validated serological clue of NAFLD is not yet available. Association of Adenovirus Ad-36 seropositivity with obesity but not with Non-Alcoholic-Fatty-Liver Disease (NAFLD) was previously shown. The aim of this study is to investigate if non-diabetic patients with Ad37+ seropositivity show different prevalence of overweight-obesity, Insulin Resistance, assessed by HOMA, higher CRP, and/or of bright liver in comparison with Ad37-seronegative patients.

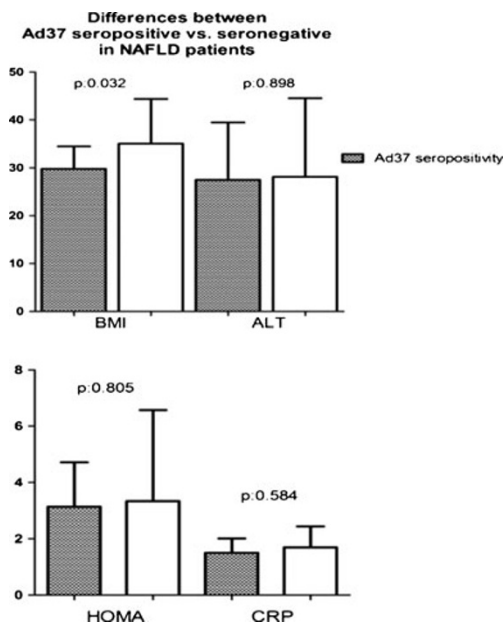
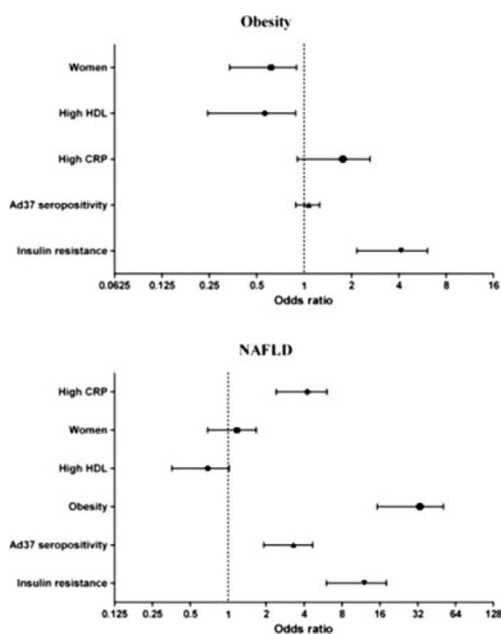
Patients and methods: 268 adult non-diabetic patients (men 146, women 122) referred for essential hypertension, overweight-obesity, and/or hyperlipidemia were treated by lower salt/lower calories Mediterranean diet, physical activity increase and smoking withdrawal counselling by the support of a health psychologist; Ad36-seropositive patients are excluded from data analysis.

Results: 65/268 patients are Ad37+ seropositive and 82/268 patients are both Ad37- and Ad36- seronegative. Obesity prevalence, defined as BMI $\geq$ 30, is not significantly different in Ad37+ (11/65; 16.9%) vs. Ad37- seronegative (15/82; 18.2%) subjects;



Bright Liver is present in 22/65 (33.8%) Ad37+ seropositive patients vs. 13/82 (15.8%) Ad37-seronegative patients ( $p<0.019$ ).

Odds Ratios show an increased hazard to Obesity associated with greater insulin resistance and CRP, while higher HDL-Cholesterol is associated with lower obesity prevalence. A more consistent hazard of Ad37 seropositivity is associated with NAFLD, along with greater insulin resistance and CRP.



### Linear regressions to BMI and HOMA

BMI						
	<i>R</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>sig</i>	$\beta$	<i>p</i>
	0,892	0,796	26,658	<0,0001		
Ad37 seropositivity					-0,215	<b>0,029</b>
FM,%					0,036	0,640
HDL Cholesterol, mg/dl					0,320	<0,0001
LDL Cholesterol, mg/dl					0,461	<0,0001
HOMA					0,952	<0,0001
CRP					-0,020	0,796

HOMA						
	<i>R</i>	<i>R</i> <sup>2</sup>	<i>F</i>	<i>sig</i>	$\beta$	<i>p</i>
	0,912	0,831	33,643	<0,0001		
Ad37 seropositivity					0,261	<b>0,003</b>
FM,%					0,015	0,831
HDL Cholesterol, mg/dl					-0,357	<0,0001
LDL Cholesterol, mg/dl					-0,406	<0,0001
CRP					-0,008	0,914
BMI, kg/m <sup>2</sup>					0,788	<0,0001

BMI: Body Mass Index; FM: Fat Mass; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; CRP: C-Reactive Protein Weighted Least Squares Regression—Weighted by Gender

Conclusion: Ad-37 seropositivity could be tentatively considered a hallmark of a clinical-metabolic profile associated with Fatty liver and NAFLD also in non-obese patients.

### MATCHING EGG COMPOSITION WITH PERSONAL MEDICAL/METABOLIC RISK: A CASE STUDY FOR A FUNCTIONAL NUTRITION APPROACH TO PPPM

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Epidemiological and clinical studies linking egg consumption to increased all-cause mortality, heart disease, and diabetes have led medical doctors to warn against egg consumption, especially in people with disease risk.



However individual predisposition and dietary factors have shown significant effect on this link, i.e. egg was found to increase plasma cholesterol mostly in genetically-predisposed ‘responders’ (33% of the population); much higher increase in CVD risk with high egg intake was found among diabetics; lack of increased LDL was noted with 2eggs/day associated with reducing diet and weight reduction. Beyond elevated plasma cholesterol and LDL levels, high egg intake increased low-density lipoprotein (LDL) oxidation (37% higher with two eggs/day vs. 2–4/week), post-prandial lipemia (with >140 mg cholesterol/meal), inflammation (elevated C-reactive protein, amyloid A, adipose-tissue macrophages), fasting blood-glucose, cardiovascular disease and all-cause mortality in diabetics (two-fold), and new-onset diabetes.

Egg modification can address most of these effects, i.e. two eggs/day with reduced n-6 polyunsaturated fatty acids (PUFA) and increased n-9 monounsaturated fatty acids (MUFA) and antioxidants (vitamin E, carotenoids, selenium) reversed two/day regular egg-induced increased LDL oxidation and fasting blood glucose to levels of 2–4 eggs/week; one/day n-3 PUFA-fortified egg increased high-density lipoprotein (HDL) levels, LDL particle size, and endothelial

function, and reduced plasma CRP, apolipoproteinB: apolipoproteinA1 ratio, triglycerides, hemoglobin A1c, and fasting glucose/glycemic response.

Personalized diagnosis of genetic/metabolic predisposition and actual state could specify individual risk and requirements. This could correspondingly direct the optimal match for egg type, preparation, and meal-planning, i.e. Greek/Mediterranean style (adding low n-6 PUFA eggs to a high n-3 PUFA, n-9 MUFA, and antioxidant meal) vs. English/butter-and-bacon (adding high n-6 ‘regular’ egg to saturated fat and red/processed meat meal). This could have additional effects and further modify egg-related physiopathological outcomes, for consumer benefits vs. risks.

Matching personalized evaluation of metabolic/genetic markers with the combined effect of egg composition/preparation and meal content, as shown by postprandial response, could best fit personal/sub-population needs and enabling informed selection and constructive expansion of egg consumption.

Most people will benefit by specified matching, though consumption may remain advised against in some metabolically/genetically-sensitive individuals.